Please amend the paragraph beginning at page 11, line 25 as follows:

C2

-- An IL-16 antagonist functions in two ways. The antagonist can compete with IL-16 for the cell surface receptor thereby interfering with, blocking or otherwise preventing the binding of IL-16 to an IL-16 receptor. This type of antagonist, i.e., which binds the receptor but does not trigger signal transduction, is also referred to herein as a "competitive antagonist" and is a feature of the present invention. Alternatively, an IL-16 antagonist can bind to or sequester IL-16 with sufficient affinity and specificity to substantially interfere with, block or otherwise prevent binding of IL-16 to an IL-16 receptor, thereby inhibiting, suppressing or causing the cessation of at least one IL-16-mediated biological activity, such as T-cell chemotaxis, for example. This type of IL-16 antagonist, also termed a "sequestering antagonist" is more specifically described in commonly-owned, co-pending (pending) application Serial No. 09/368,632, filed on August 5, 1999 and entitled "IL-16 Antagonists", the teachings of which are incorporated herein by reference.--

Please amend the paragraph beginning at page 30, line 12 as follows:

C3

--Human peripheral blood mononuclear cells (PBMC) were isolated as described (Center, et al., *J. Immunol.* 128:256, 1982; Cruikshank, et al., *J. Immunol.* 128:2569, 1982 and Cruikshank, et al., *J. Immunol.* 138:3817, 1987) from the blood of healthy volunteers by density centrifugation on Ficoll-Paque reagent (Pharmacia, Piscataway, NJ). The mononuclear cell layer was washed with medium 199 (M.A. Bioproducts, Walkersville, MD) supplemented with 0.4% bovine serum albumin, 25 mM HEPES buffer, and 100 U/ml of penicillin and 100 µg/ml streptomycin (M199-HPS). Samples were enriched for T lymphocytes by nylon wool adherence as described (Julius, et al., *Eur. J. Immunol.* 3:645, 1973). The nonadherent cells were >95% $CD3^+$ as determined by flow cytometry.--